



## Case report

### Unusual case of right atrial reinfarction

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#### ARTICLE INFO

##### Article history:

Received 9 May 2011

Received in revised form

13 August 2011

Accepted 3 October 2011

Available online 28 October 2011

##### Keywords:

Atrial reinfarction

Atrial infarction

Atrial ECG changes

#### ABSTRACT

It is well known that atrial infarctions are rare comparing to the ventricular. They cannot easily be verified on ECG and the standard autopsy technique does not include a detailed review of the atrial wall, so the atrial infarction often remains undiagnosed. A 63-year-old male was treated and died in an intensive care unit due to decompensated liver insufficiency and cardiac disease following long-lasting alcohol abuse. At autopsy, the extreme cardiomegaly was found, severe atherosclerosis of the anterior descending branch of left coronary artery. The posterior wall of the right atrium was thickened (cca 9 mm) in diameter of cca 3 × 3 cm, and this area was yellowish in the luminal part, while the central part was filled with dark red blood. A detailed dissection of the coronary arteries showed the complete occlusion of the atrial branch of the right coronary artery wreath as far as the place of sinoatrial artery branching, which corresponded anatomically to the described area of infarction on the posterior wall of the right atrium. Histopathological examination of the previously described area of the posterior wall of the right atrium, showed four zones of heart muscle changes: 1. zone of partially preserved structure of the heart muscle, 2. zone of cellular (immature) connective tissue, 3. areas of bleeding in cellular connective tissue, and 4. zone of acellular (old) connective tissue. These histopathological changes indicated that the posterior wall of the right atrium was affected by myocardial necrosis in at least two and possibly more times. It is reasonable to think that bleeding in the third zone of the posterior wall of the right atrium contributed greatly to the death due to the anatomical proximity to the sinoatrial node. It was confirmed by the existence of bradycardia with a prolonged PR interval, PR segment elevation in D1 and aVL lead and PR depression in the D3 lead on the ECG. These ECG changes appeared immediately before asystolia and the death of the patient, but not ventricular fibrillation or electromechanical dissociation due to ventricular infarction. The presented case shows that detailed autopsy examination of atrial wall and blood vessels can sometimes be crucial in disclosing the cause and mode of death if the ischemia and necrosis attack only the atrial wall, especially in the region of the heart conduction system.

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## 1. Introduction

Cardiovascular diseases, particularly ischaemic heart disease, have been written about more than any other medical entity (about 1.7 million articles in PubMed) save for malignancies (about 2.5 million articles). Not only are they the leading cause of death and behind a significant amount of expenditure on health insurance, but, despite extensive preventive propaganda, it seems that cardiovascular diseases are also a global social problem that will be difficult to solve.

Atherosclerotic plaque rupture, thrombo-embolism and vaso-spasm are the most common initiators of clinical symptoms of

myocardial ischaemia, which often results in necrosis – a heart infarction. Myocardial infarction can be easily confirmed by well-known electrocardiogram (ECG) changes and by elevated cardiac enzymes. During autopsy, the acute myocardial infarction is macroscopically verified as a pale area, but only if the duration of the ischaemia is more than a few hours, usually more than six. The first histopathological signs of acute ischaemia may occur after only 30 min to 4 h as waviness of the peripheral myofibrils, oedema of the myofibrils and the increased presence of neutrophils in the blood vessels. Later signs of necrosis of cardiac myocytes are eosinophilia, loss of horizontal striping and the presence of neutrophils in the interstitium, while finding hypercontraction bands is a little bit controversial, since it can also be found in different kinds of increased catecholamine activity (e.g., the injection of adrenalin during cardiopulmonary resuscitation (CPR)). Fields of fibrosis represent a scar on a former zone of necrosis.

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Infarctions of the left ventricle and interventricular septum dominate, while isolated right ventricular infarctions are present in just 1–3% of cases.<sup>1</sup>

The first case of atrial infarction was described in 1925, while later research has shown a frequency ranging from 0.7% in ante-mortem up to 42% in autopsy studies.<sup>2</sup> Due to the large number of anastomotic branches of the coronary arteries, as well as the presence of oxygenated blood in the cavity of the left atrium, right atrial infarctions are four to five times more prevalent than those in the left atrium.<sup>3,4</sup> They cannot easily be verified on ECG and some of the changes combine elevation and depression of PT segments, often accompanied by atrial arrhythmias (flutter and fibrillation), a prolonged PR interval and abnormal Q-wave and P-wave axis changes.<sup>2,5–12</sup> Despite these facts, the standard autopsy technique does not include a detailed review of the atrial wall, so the atrial infarction often remains undiagnosed.

## 2. Case report

A 63-year-old male was treated and died in an intensive care unit due to a severe general health condition, disorientation, psychomotor weakness, fever ( $38.4^{\circ}\text{C}$ ) and dyspnoea, all explained as signs of decompensated liver insufficiency due to cirrhosis, hepatic encephalopathy, jaundice and ascites, following long-lasting alcohol abuse. Elevated values of direct bilirubin ( $85.2 \text{ mmol l}^{-1}$ ), total bilirubin ( $140.0 \text{ mmol l}^{-1}$ ) and urea ( $8.9 \text{ mmol l}^{-1}$ ) were verified. ECG changes were in favour of acute ischaemia of the anterior wall of the left ventricle and the interventricular septum.

External examination of the corpse revealed that he was an obese person with yellowish skin and visible mucous membranes, trophic skin changes and doughy oedema of the legs and fluctuation of the anterior abdominal wall. The autopsy revealed a brain oedema, a pulmonary oedema with 1000 ml of pleural effusion, oesophageal varices, almost a massive 14 l of ascites fluid, micronodular cirrhosis of the liver, splenomegaly (410 g), nephrosclerosis and 700 ml of pericardial effusion.

A detailed examination of the heart, which was  $19 \times 20 \text{ cm}$  in diameter and weighed 1050 g (Fig. 1), showed blotchy areas of haemorrhage and whitish fibrosis of the epicardium, diffuse fibrosis of the left ventricular endocardium and dotted endocardial haemorrhage of the right atrium. The anterior descending branch of left coronary artery was completely calcified, appearing like a brittle wire, while the intima of the right coronary artery was mostly smooth, with sparse atherosclerotic plaques. On the cut surface of the heart muscle, diffuse fibrosis was noticed in the form of small whitish strips, particularly in the area of the papillary muscles, and two pale zones, one of which included almost the entire anterior wall of the left ventricle and ventricular septum, and the other the middle third of the diaphragmatic wall of the left ventricle (Fig. 2).

One yellowish thickening of the posterior wall of the right atrium of cca  $3 \times 3 \text{ cm}$  in diameter was also observed, which aroused suspicion of a heart infarction in that region, the thickened wall (cca 9 mm) was examined by cutting and was yellowish in the luminal part, while the central part was filled with dark red blood (Fig. 3). After a formaline fixation, a detailed dissection of the coronary arteries showed the complete occlusion of the atrial branch of the right coronary artery wreath as far as the place of sinoatrial artery branching, which corresponded anatomically to the described area of infarction on the posterior wall of the right atrium (Fig. 4).

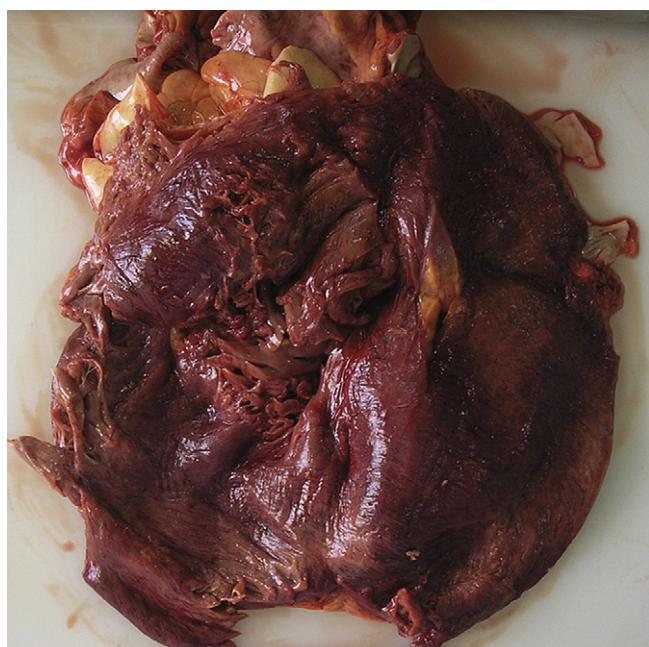
Histopathological examination (haematoxylin–eosin staining) confirmed chronic ischaemic heart disease with diffuse interstitial fibrosis, fields of cardiomyocyte attenuation and undulation, of



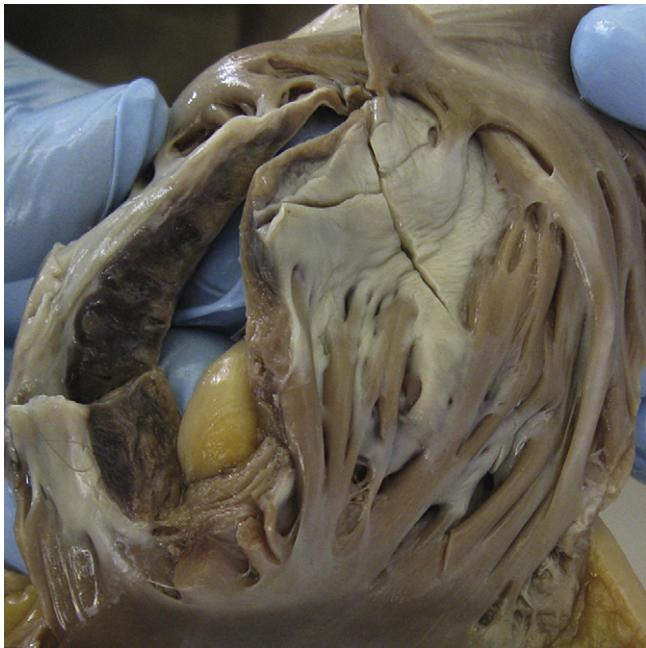
**Fig. 1.** Dilatative cardiomyopathy.

which some lost the transverse striping, and many area of immature and older connective tissue. In the previously described area of the posterior wall of the right atrium, four zones of heart muscle were clearly distinguished, viewing from the outside to the luminal side (Fig. 5):

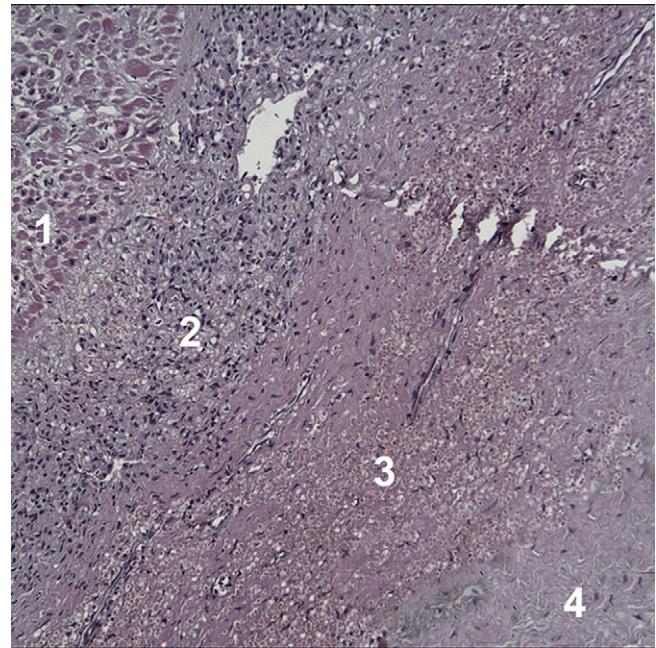
1. zone of partially preserved structure of the heart muscle,
2. zone of cellular (immature) connective tissue,
3. areas of bleeding in cellular connective tissue and
4. zone of acellular (old) connective tissue.



**Fig. 2.** Pale zones on the section of left ventricle.



**Fig. 3.** Posterior wall of right atrium.



**Fig. 5.** Posterior wall of right atrium (HE, x10).

### 3. Discussion

In the presented case, the cardiac disease and hepatic complications associated with chronic alcohol abuse led to death. Their individual contribution is difficult to evaluate, but their synergistic or potentiating effect was evidently fatal.

Although the history of heart problems could not be considered based on the available medical records and the data obtained from relatives, it was clear from the established histopathological changes that dilated cardiomyopathy, among other things, produced the long-lasting repetitive anginal symptoms. The

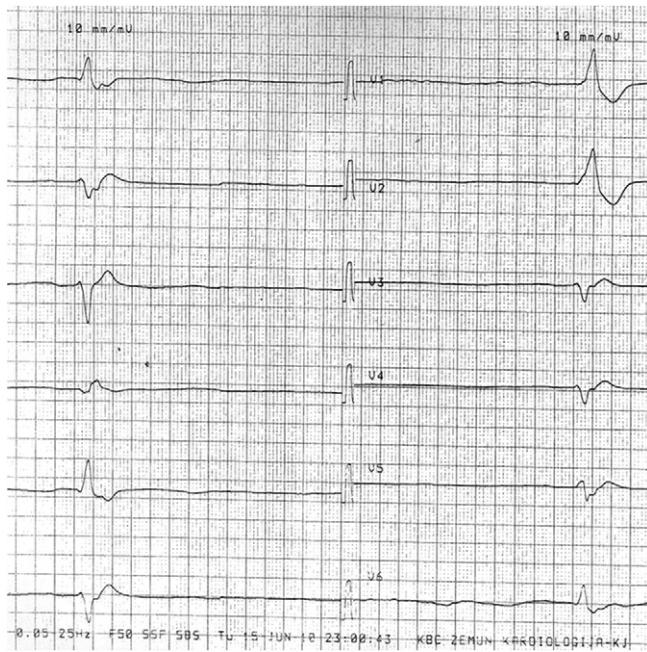
repetitive attacks of ischaemia produced numerous changes in the form of small myocardial infarctions, while in at least two and possibly more times, the posterior wall of the right atrium was affected by myocardial necrosis. This was concluded due to the presence of infarction zones of different ages (Fig. 5).

The zone marked with the number 4 represents the area of fully organised scar tissue composed of collagen fibres and sparse fibrocytes (an at least 6-week-old infarction). Zones 2 and 3 are formed of immature connective tissue with an abundance of cellular elements and less collagen fibres, representing a zone that occurred recently – that is, in a second necrotic attack, with abundant haemorrhage in zone 3. Along with the large fields of acute left ventricular ischaemia and multi-organ failure among hepatic insufficiency, which were probably the main causes of death, it is reasonable to think that bleeding in the third zone of the posterior wall of the right atrium contributed greatly to the death due to the anatomical proximity to the sinoatrial node. It was confirmed by the existence of bradycardia with a prolonged PR interval, PR segment elevation in D1 and aVL lead and PR depression in the D3 lead on the ECG (Fig. 6). These ECG changes appeared immediately before asystolia and the death of the patient, but not ventricular fibrillation or electromechanical dissociation due to ventricular infarction.

Circa 10% of ventricular infarcts are followed by atrial infarct, or at least they are suspected. Usual ECG changes in atrial infarcts are manifested by changing of rhythm or changing in PR, PQ or Pta (P-T atriale) segment. As we mentioned in introduction, there are so many atrial infarction criteria that any of dysrhythmias originated by atria can be recognised as their infarct, as well as Pta elevation ( $>0.5$  mm) in I lead accompanied by Pta depression in II and III, or Pta depression ( $>1.5$  mm) in precordial leads. Nevertheless, in literature, many other suggestions and criteria for atrial infarct verification could be found, dealing with different combinations of ECG changing and atrial arrhythmias.<sup>2,5,10–12</sup> Concerning these facts, ECG changing and bradycardia found in the presented case report can also be easily recognised as atrial infarction, but its real contribution to death remains a dilemma.



**Fig. 4.** Atrial branch of right coronary artery.



**Fig. 6.** ECG ante finem.

#### 4. Conclusions

Finally, the question of the expediency of searching for atrial infarcts during autopsy is posed. There is no doubt that the exclusion of other possible causes of death and verification of myocardial ischaemia in the area of the ventricular walls certainly leads to a conclusion of natural cardiac death. However, dutiful and detailed autopsy work surely includes the section of atrial walls, which contributes to the quality of the work, gives a more detailed insight into chronic ischaemic heart disease and can sometimes be crucial in disclosing the cause and mode of death if the ischaemia and

necrosis attack only the atrial wall, especially in the region of the heart conduction system.

#### Conflict of interest

Authors havn't any financial benefits on publishing the paper.

#### Funding

None.

#### Ethical approval

None.

#### References

- Schoen FJ, Mitchell RN. The heart. In: Kumar V, Abbas AK, Fausto N, Mitchell RM, editors. *Robbins basic pathology*. 8th ed. Philadelphia: Saunders Elsevier; 2007. p. 391–4.
- Shakir DK, Arafa SOE. Right atrial infarction, atrial arrhythmia and inferior myocardial infarction form a missed triad: a case report and review of the literature. *Can J Cardiol* 2007;23(12):995–7.
- Gardin JM, Singer DH. Atrial infarction. Importance, diagnosis and localization. *Arch Intern Med* 1981;141:1345–8.
- Lazar EJ, Goldberger J, Peled H, Sherman M, Frishman WH. Atrial infarction: diagnosis and management. *Am Heart J* 1988;116:1058–63.
- Liu CK, Greenspan G, Piccirillo RT. Atrial infarction of the heart. *Circulation* 1961;23:331–8.
- Wong AK, Marais HJ, Jutzy K, Capestany GA, Marais GE. Isolated atrial infarction in a patients with single vessel disease of the sinus node artery. *Chest* 1991;100:255–6.
- Rose KL, Collins KA. Left atrial infarction: a case report and review of the literature. *Am J Forensic Med Pathol* 2010;31:1–3.
- Neven K, Crijns H, Gorgels A. Atrial infarction: a neglected electrocardiographic sign with important clinical implications. *J Cardiovasc Electrophysiol* 2003;14:306–8.
- Di Maio V, Di Maio D. *Forensic medicine*. 2nd ed. London – New York: CRC Press; 2001.
- Jim MH, Miu R, Siu CW. PR-segment elevation in inferior leads: an atypical electrocardiographic sign of atrial infarction. *J Invasive Cardiol* 2004 Apr;16(4):219–21.
- Nielsen FE, Andersen HH, Gram-Hansen P, Sørensen HT, Klausen IC. The relationship between ECG signs of atrial infarction and the development of supraventricular arrhythmias in patients with acute myocardial infarction. *Am Heart J* 1992 Jan;123(1):69–72.
- Maganis JC, Gupta B, Gamie SH, LaBarbera JJ, Startt-Selvester RH, Ellestad MH. Usefulness of p-wave duration to identify myocardial ischemia during exercise testing. *Am J Cardiol* 2010 May 15;105(10):1365–70. Epub 2010 Apr 2.